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Alternate Hosts of *B. Vasculorum*,

The Causal Agent of Gumming Disease of Sugar Cane,

by

C. G. HUGHES.

Issued by Direction of the Hon. F. W. BULCOCK,
Secretary for Agriculture and Stock.

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C. G. Hughes.

The serious disease of sugar cane caused by *Bacterium vasculorum* (Cobb) Greig-Smith has been known under various names: "Maladie de la Gomme," "Gummosis of the Sugar Cane," "Cobb's Disease of Sugar Cane," and "Gum Disease" are all synonyms. Generally, however, the term "gumming disease" is now preferred.

GUMMING DISEASE.

Historical.

Gumming is one of the oldest recorded diseases of sugar cane. Some 70 years ago Dränert [**8**, 1869] described a disease which at that time was widespread in the sugar-cane crops of Brazil. An examination of his published description of symptoms leaves little doubt that he was dealing with gumming, and circumstances strongly suggest the western hemisphere as the place of origin of this disease. Its extension into the eastern hemisphere appears to have followed a transfer of cane varieties from Brazil to Mauritius in 1869 (Bonâme [**4**] 1894). It was recorded as being present in Madeira in 1883, and since that time has been authentically recorded in Australia, Fiji, Puerto Rico, St. Kitts, St. Lucia, Colombia, Dominica, Antigua, Guadeloupe and Barbados (*see* Bell [**3**] 1929 and North [**14**] 1935). Mosaic alone among the major diseases of sugar cane has a wider geographical range.

Precise data regarding the introduction of gumming disease into Australia are not available, but it was certainly present in the Clarence River district of New South Wales in 1892 ("Agricola" [**1**] 1892). Prior to this date there had been several recorded importations of cane plants from Mauritius, and there is no doubt that there were numerous unrecorded private introductions also, and so, having in mind the nature of the disease, we may fairly safely assume that it was introduced from Mauritius in one or more of these shipments. It is suggested from the writings of North [**14**, 1935], Cobb [**6**, 1893] and Tryon [**18**, 1895], that it was present in New South Wales some few years prior to its establishment in Queensland, although it was apparently present in the Mackay district at least as early as 1890 (Knox [**12**] 1894). It may have been introduced into Queensland by plants from New South Wales, as exchange of plants was common, but the records of the Colonial Botanist also show that several importations were made direct into Queensland from Mauritius about this period.

A detailed study of the disease, with the identification of the causal organism, was made by Cobb [**6**, 1893] during a period of service as Pathologist

to the New South Wales Government. His etiological studies were later extended and confirmed by Greig-Smith [**9**, 1902], and Erwin F. Smith [**17**, 1914]. In more recent times a great deal of additional investigation has been carried out in New South Wales on behalf of the Colonial Sugar Refining Coy. Ltd. by North [**14**, 1935].

The first recorded investigation of the disease in Queensland was undertaken by Tryon [**18**, 1895], the then Entomologist and Vegetable Pathologist of the Department of Agriculture. Tryon commenced his investigations in the Wide Bay and Burnett districts in October, 1894, following outbreaks of the disease in the winter of that year. He states that "there is abundant evidence to show that it (gumming disease) was no novelty, although formerly it neither occurred so widely nor was so virulent in its attacks as subsequently was the case." He concluded from his inquiries among planters that the disease had been present in the Wide Bay district at least ten years previously. The possible presence of the disease in the Mackay district in 1890 is mentioned above.

Following this epiphytotic of the 'nineties, gumming disease was gradually brought under control in the southern district by trial and error substitution of more resistant varieties. The disease declined in importance in succeeding years, becoming virtually extinct in the period 1915-1920, during which time the proportion of susceptible varieties cropped rose to 98 per cent. (Bell [**2**] 1936). A further outbreak became evident during 1920-22, and reached a peak in 1929, but has since been brought entirely under control by the systematic substitution of resistant varieties (*see* later).

Sporadic outbreaks have occurred from time to time in the central (Mackay) district but, in spite of the cultivation of a large proportion of varieties which are highly susceptible in other districts, epiphytotics do not occur. The reason for this fortunate circumstance presumably lies in the particular environmental conditions, but it must be admitted that the specific controlling factors are not known. In the northern (wet tropical) district outbreaks have occurred in two centres, viz., Herbert River District and Mulgrave. In the former the disease first made its appearance in the early years of this century, to be followed some ten-fifteen years later by a widespread outbreak which, however, has since been brought under complete control by systematic planting of resistant varieties. A small outbreak was discovered in the Mulgrave area early in 1925; after a few years the disease appeared to have been eradicated, but in 1934, following widespread plantings of the very susceptible S.J.4, a recrudescence occurred. It is uncertain whether this was due to a fresh introduction of diseased cane or to the persistence of a local focus of infection. In any case this occurrence points to the necessity for caution in planning a return to susceptible varieties in a once infected locality.

The Queensland cane lands extend north-south in a broken chain along 1,100 miles of coast line, and, as would be supposed, such a range of latitude embraces very considerable variations in climate and soil conditions, leading to wide variations in the severity of the disease in different districts. It is in the southern districts about Bundaberg that the disease has been responsible

for most damage, and it was in this area that the Division of Pathology in the Bureau of Sugar Experiment Stations initiated systematic gumming resistance trials in 1928. During the course of the next few years hundreds of varieties, both importations and the Bureau's own seedlings, were tested. Satisfactory resistant varieties were found in Co. 290, P.O.J. 213, P.O.J. 234, P.O.J. 2725 and P.O.J. 2878; with the almost exclusive cultivation of these, gumming is rapidly becoming rare and has ceased to be a direct economic factor in the south.

The question now arises as to whether it will be safe to plan a return to susceptible varieties. In this regard North [**13**, 1932] and [**14**, 1935] has struck an optimistic note in asserting that gum has a peculiar tendency to become extinct when resistant varieties are grown; certainly this appears to have been the case in Fiji where plantings were under complete control. However, extinction of a disease may be more apparent than real, since it is possible for the causal organism to persist in moderately resistant plants, or in isolated and unnoticed susceptible individuals, just waiting an opportunity, as it were, to infect a susceptible variety which may hopefully be introduced on a large scale. The happenings in the Mulgrave area indicate the necessity for extreme caution under the small farm system, although with adequate variety control such a plan should by no means be considered impracticable unless, of course, cane is not the only natural host of *B. vasculorum,* when the question would require re-examination. The possible existence of alternate hosts appeared to us to be of sufficient importance to warrant investigation. We have already directed attention (p. 35) to the fact that *B. vasculorum* appears to have first attacked cane in the western hemisphere, where sugar cane is not indigenous (Brandes & Sartoris [**5**] 1937); such a combination of circumstances is, in our opinion, *prima facie* evidence in favour of the existence of at least one alternate host.

B. Vasculorum in Sugar Cane.

Bacterium vasculorum is a short rod-shaped bacterium (*see* Fig. 1) 1·0-1·5*u* long by 0·4-0·5 diameter, motile by means of a single polar flagellum, occurring singly, in pairs, or in short chains. It does not form spores, but in the mass may be capable of considerable resistance to unfavourable environmental conditions, due to the protective action of the "gum."

Symptoms of Gumming Disease.

The symptoms of gumming disease, according to the standard description adopted in this laboratory, are as follows:—

Gumming disease is primarily a leaf disease and when contracted by resistant varieties does not normally progress beyond this stage but, in the case of susceptible varieties, it may pass down into the stem and become systemic. The earliest symptom exhibited by the plant, and the most convenient for field diagnosis of the disease, is the presence of characteristically coloured longitudinal streaks upon the leaves (*see* Plate 1). The streaks follow the course of the vascular bundles and thus run straight and at a slight angle to

the midrib. They are generally uniform in width and 3-6 mm. wide; the margins are fairly well defined but older streaks become diffuse and lose some of their regularity of outline. They may arise in any portion of the leaf blade, but the majority arise near the margin and towards the apex; the length varies from a few millimetres to almost the full length of the leaf. The colour of the streaks is yellow to orange, usually flecked with large numbers of reddish dots. At the point of origin of the older streaks the tissue dies and becomes ashy coloured, the dead area gradually extending longitudinally in both directions if the streak has arisen in the middle of the leaf, or inwards if the streak has commenced at the margin. Streaks of this type are not found

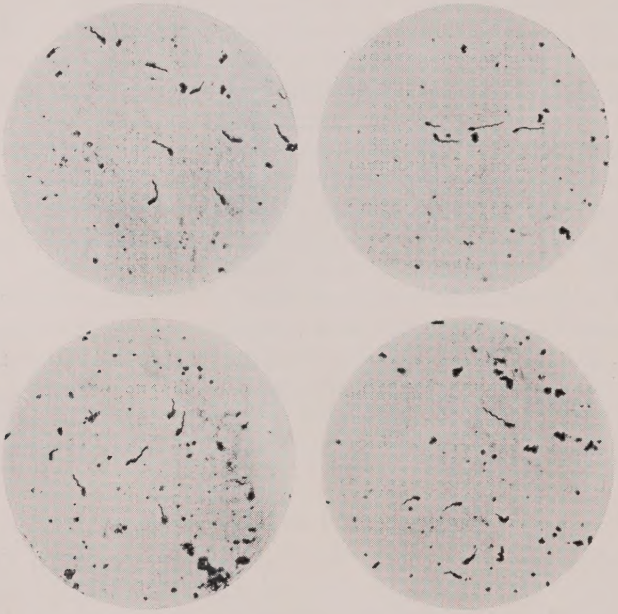

FIG. 1.—Photomicrographs of *B. vasculorum*, showing flagella. Material from agar slope of Wilbrink's medium, centrifuged in water to remove gum, stained by Bailey's method.

on the youngest leaves, are confined to the leaf blades, and there may be one to several on any one leaf. They are most readily found some two or three weeks after periods of wet weather, but may be very hard to find after prolonged dry weather, especially in young cane.

In the final stages of the disease, following systemic infection, reddish leaf streaks, similar to those of red stripe (*see* Cottrell-Dormer [**7**] 1932), may be found in certain varieties, particularly at the base of the leaves in the cornucopia, whence they pass down to the stem. Certain other susceptible varieties (e.g., P.O.J. 2714) show an additional symptom in the more or less frequent production of creamy-white streaks, very similar to the early diffuse stage of streaks of leaf-scald disease (Bell [**2**] 1929). The production of

PLATE 1.—Leaf symptoms of gumming disease in sugar cane. Typical streaks are yellow to orange in colour, flecked with numerous reddish dots, later developing ash-coloured areas of dead tissue. The outline of the streaks is at first fairly regular, but later becomes diffuse.

chlorotic tops is another leaf-borne symptom often following systemic infection; one to all leaves may be involved, and this condition also does not usually long precede death.

The outstanding characteristic of this disease, and the one formerly used almost exclusively for diagnosis, is the exudation of a yellowish to orange-red gummy mass from the cut ends of badly diseased stems (*see* Fig. 2). With canes which are not so badly diseased it is often possible to induce the gum to flow by placing a piece of freshly cut stalk in a small closed container at 35-40°C.; in the presence of very light systemic infection, however, it may be necessary to cut the cane into setts and plant in moist sand, keeping the temperature at about 30°C., dig up after a period of 4-6 weeks, cut fresh surfaces, and sweat as above. Small droplets of gum may also be sweated from the freshly cut ends of turgid leaf-streak tissue placed in a petri dish or suitable small closed container; similar exudation may take place in the field, during wet weather, from wounds in leaf-streak tissue. The exudate, or so-called gum, when examined under the microscope is found to be composed of countless rod-shaped bacteria and an amorphous gummy substance. The bacteria are confined mainly to the elements of the xylem in the stem and leaves and the resultant gum causes a clogging of these vessels.

Frequently, and especially in the region of the growing tip, the bacteria may invade the parenchyma surrounding the vascular bundles and cause a rapid break-down of the tissues, giving rise to "gum pockets." The latter may be seen when a badly diseased cane is split longitudinally; they consist of cavities of varying size, filled with a gummy mass, and sometimes containing as much as a cubic centimetre of the gum. It has been found that these represent practically pure cultures of the organism.

Reddened vascular bundles, especially at the nodes, are a usual feature of gumming disease, but this symptom is common to a number of diseases and hence of little use as a specific diagnostic aid, although most useful for indicating the parts from which gum may be sweated. As a rule the typical leaf symptoms can readily be found long before any macroscopic manifestations of the disease are apparent within the stem tissues; an exception is the case of varieties in which the leaf symptoms are insignificant and transient but in which systemic infection may develop under suitable conditions.

Stunting of the plant usually follows systemic infection, the upper internodes tapering off almost to a point. The death of the growing point may cause the lateral buds to develop and give rise to side shoots, though this latter feature is not pronounced. When growing conditions are favourable there may be little death of cane caused by the disease, but during periods of dry or excessively wet conditions the badly affected plants wilt and eventually develop a dry top rot and die. In some varieties this wilting phase may be very rapid. In the final stages large discoloured cavities may be found running down the centre of the stem. With susceptible varieties, such as S.J.4, the losses due to the death of cane may be as high as 100 per cent. In general, the incidence

FIG. 2.—Exudation of "gum" from the freshly cut end of diseased cane (variety P.O.J. 2714) after "sweating" in a sealed container.

of the disease is much lighter in ratoon crops than in plant cane, but in the case of a badly diseased susceptible variety many stools may fail to ratoon or give rise to weak chlorotic shoots, the majority of which soon die out.

Transmission.

Many agencies may be involved in the spread of gumming from field to field or farm to farm; knives unsterilized after use in a diseased crop are important from this point of view since *B. vasculorum* can survive even on a dry surface for a number of days (North [**14**] 1935). Agricultural implements, transport vehicles, farm workers, and animals may carry inoculum over quite long distances. Diseased cane or cane tops in transit might infect growing plants by brushing against them, while insects can carry the disease to fresh fields. However, diseased planting material is the most important agency in the long distance transmission of gum; setts outwardly healthy may yet contain the organism and, giving rise to diseased stools, infect a new area.

Within the field the disease may be disseminated by any of the methods listed above but combined wind and rain constitute by far the most important agency in intra-field spread. In windy weather the leaves of the cane plants are blown about and rub together so that the sharp serrated edges cut and scratch the blades of adjoining leaves; rain drops falling on gummy exudate or on exposed disease tissues, such as torn streaked leaves, will dissolve some of the gum and then be blown or shaken on to the leaf wounds and so set up fresh infection. If the leaves are reasonably upright as in Q.2, D.1135, and S.J.4 the serrations cut the edges of the leaves in such a way that the point of infection is usually close to the tip. On the other hand, in varieties like Black Innes and M.1900 Seedling, in which the leaves droop, the first infection usually occurs at a point somewhere on the bend of the blade.

Artificial Inoculation.

The usual experimental method of inoculation of growing cane plants consists in piercing the heart of the cabbage several times with a bundle of needles smeared with a suspension of the organism. Cotton wool wrapped around the needles (leaving only the tips exposed) and dipped in a suspension of the organism forms a convenient reservoir of inoculum sufficient for the inoculation of several shoots. Leaf streaks appear after some two weeks if conditions are favourable. Infection may also be induced in cane by injecting a suspension of the organism into the stick or the growing point, or by removing a small core of tissue from the stem with a fine cork borer, pouring in a little bacterial suspension, and re-sealing the hole.

Although the various methods outlined above are quite effective for the inoculation of cane they would cause considerable damage to the tissues of a plant less robust, and so, in carrying out inoculations of other grasses, several refinements of apparatus were introduced (*q.v.*).

ALTERNATE HOSTS OF *B. VASCULORUM.*

Review of Literature.

Although trials (Bell [**2**] 1928-37) have shown that gumming disease will infect varieties of the species *Saccharum robustum, S. sinense, S. spontaneum,* and *S. officinarum* as well as hybrids, until recently no attempt had been made to ascertain whether other members of the family Gramineæ could be infected. Ivanoff [**10**, 1933] and [**11**, 1935] appears to have been the first worker in this field. He recorded the infection of corn, *Zea mays* L. and sorghum, *Holcus sorghum* L. with *B. vasculorum,* using cultures obtained from this Bureau. This work was incidental to host range investigations with the causal agent of Stewart's wilt disease of corn, *Phytomonas stewarti* (E. F. Smith) Bergey *et al.,* the similarity of symptoms and of growth in culture of the two organisms suggesting that there may be a common host range. The symptoms induced in corn by *B. vasculorum* were described as being of three types: water-soaked stripes along infected leaf veins, cavities in the stalks and general stunting. The varieties of corn used were Golden Gem and Golden Bantam, both sweet corns known to be very susceptible to Stewart's disease. Dent corn varieties were not inoculated.

Ivanoff also attempted reciprocal inoculation of sugar cane with cultures of *P. stewarti* but failed to obtain positive results. In this connection, however, it should be noted that the varieties used, viz., Co.281 and C.P.807, are both highly resistant to gumming disease. Poos and Elliott [**16**, 1936] likewise record failure to obtain infection of "two varieties of sugar cane" with Stewart's disease, but they do not record the identity of the varieties.

We have observed in the "Review of Applied Mycology" an abstract of a paper by G. Orian [**15**, 1937]. The author is stated to record the isolation of *B. vasculorum* from a heart rot of white palms (*Dictyosperma album*) and reisolation from plants of the same host and maize. The reisolates were identified at the Imperial Mycological Institute and on inoculation into sugar cane in Mauritius produced the characteristic symptoms of gumming disease. It is noted that a yellowish gum was exuded from the vascular bundles of the trunks of diseased palms.

Preliminary investigations on the subject of alternate hosts of *B. vasculorum* were commenced by the writer in 1935. Successful inoculation of various types of corn with *B. vasculorum* and reisolation of the pathogene without loss of pathogenicity were briefly recorded in annual reports of the work of the Division of Pathology for 1936 and 1937, Bell [**2**]. Stewart's disease of corn has not been recorded in Australia and consequently inoculations with *P. stewarti* were not attempted.

Experimental Methods and Procedure.

General.

All work was carried out in Brisbane during the spring and summer months of the seasons 1935-8. Seeds of the various varieties of corn (*Zea mays* L.) and sorghum (*Sorghum* sp. Snow) and one variety of Soudan

grass (*Sorghum sudanense* Stapf) were sown in the pathology plot in hills and the resultant plants thinned to three per hill. Stools of *Sorghum verticilliflorum* Stapf, Johnson grass (*S. halepense* (Linn.) Pers.), Para grass (*Brachiaria mutica* (Forsk.) Stapf), elephant grass (*Pennisetum purpureum* Schum.) and Guinea grass (*Panicum maximum* Jacq.) were established from cuttings or divided stools.

One type of inoculation only was carried out in any one hill or stool. The plants were irrigated when necessary by flooding. Protection from winds was obtained by the erection of hessian barriers some six feet high. This protection from wind and the use of ground irrigation gave conditions which are rather unfavourable for the natural spread of gumming disease from cane plant to cane plant; it was presumed that the same would hold for other plants, and the many inoculated plants with the necessary controls were maintained in a comparatively small area without the occurrence of a single case of natural spread.

Cultures of *B. vasculorum* on Wilbrink's medium* vary considerably in colour, vigour and consistency and fixed variants may be readily isolated and maintained. Since cultures differ in such physical features it is not unreasonable to suspect (although we have no definite evidence on this point) that other characters not so easily observable may also vary. Cultures then might vary in pathogenicity, and in order to minimise the risk of negative results through using a weakly pathogenic strain, mixtures of several cultures were used in the experiments described in this paper.

The inoculum was a suspension formed by mixing "gum" from five to ten day-old cultures on Wilbrink's agar slopes with approximately ten times its volume of sterile distilled water. This gave a slightly viscous liquid which, however, could easily be forced through the hypodermic needle used.

Controls in all inoculation experiments were treated in exactly the same way as the inoculated plants, with the substitution of distilled water for the suspension of organisms. Controls were about equal in number to the inoculated plants.

Isolates resembling *B. vasculorum,* obtained from lesions resulting from inoculations made during the course of the experiments described below, were positively identified by the use of a method detailed by North [**14**, 1935] and considered by him to be practically infallible. This is a so-called "sweating" test, and consists essentially of smearing the ends of freshly cut, fairly long setts of cane with a suspension of the particular culture and then planting in damp sand. Good germination conditions are maintained, and at the end of a month to six weeks after planting the setts are dug up and, the rotted ends having been removed, they are sweated in a warm, moist chamber for 24 hours. Droplets of gum exuding from the cut ends indicate a positive reaction.

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* Sucrose .. .. 20 grm.
Peptone .. .. 5 grm.
K_2HPO_4 .. .. ·5 grm.
$MgSO_4.7H_2O$ .. ·25 grm.
Agar .. .. 20 grm.
H_2O .. .. 1,000 grm.
Adjust pH to 6·8-7·0 with KOH.

Methods of Inoculation.

1. *Heart Inoculation.*

(*a*) Needle puncture (plants four weeks old). A drop of suspension was placed in the youngest exposed leaf-blade angle and a fine, sharp needle (Milward and Sons' No. 10 sewing needle) pricked several times through the drop into the tissues of the shoot. This technique was evolved as a refinement of the method which has proved to be quite satisfactory for the inoculation of cane varieties (*q.v.* above). Shoots of three cane varieties, viz., M. 1900 Seedling, P.O.J. 2714 and D. 1135—all known to be susceptible to gumming—were inoculated in the same way with the same inoculum as a check on pathogenicity.

(*b*) Hypodermic needle injection (plants five to seven weeks old). The ordinary hypodermic needle was found to be too coarse for work of this nature, and the method of attachment of the needle to the injector was not suitable for pressure inoculation. Recourse was had then to a fine, 27-gauge needle of the "Cartridge" type, as used by dentists. This needle was 6 cm. long and sharp at both ends; a metal collar, acting as a stop to prevent the needle being pushed back into the barrel, was fixed about 1·5 cm. from the end. The needle was attached to the injector by pushing it, short end first, through the needle holder of the injector and thence through a solid rubber washer which sealed the base of the barrel so that some 2 mm. of needle projected into the barrel cavity and the metal collar pressed hard against the needle holder. This modified injector proved to be quite satisfactory for pressure inoculation when fitted with a screw-adjustable plunger and the needle did not break nor, providing the inoculum was under slight pressure, clog when penetrations were made into plant tissues.

Inoculations were made as near as possible to the growing point of the shoot, and approximately 0·2 ml. of inoculum injected. It was found that considerable pressure was required for inoculation into the heart; if the needle point did not penetrate the stem tissue droplets of suspension merely escaped between the leaf sheaths. Approximately 20 seconds elapsed before the 0·2 ml. was absorbed into the stem tissues. It was found desirable to include an air bubble when filling the injector; then, on pushing the plunger down to give the desired dosage, the inoculum entered the stem under gradual pneumatic pressure rather than sudden direct hydraulic pressure.

2. *Leaf Inoculations.*

(*a*) Needle puncture (plants five weeks old). A drop of the standard bacterial suspension was placed on the surface of the leaf and the underlying tissue pricked through it with a very fine, sharp needle. Some 40-50 punctures were made over an area of less than half a square centimetre. Several inoculations were made on each leaf, care being taken not to inoculate the same veins or sets of veins more than once. North [**14**, 1935], using this method on cane leaves, obtained a high percentage of positive results.

(*b*) As in (*a*), with the substitution of light scratching with a fine needle for pricking.

3. *Stem or Cob-shank Inoculation.*

Some 0·2-0·3 ml. of the suspension was injected with the hypodermic needle either into the solid stem approximately 10 cm. below the growing point or into the base of young cob shanks. The plants were approximately nine weeks old when inoculated and from 6 inches to 2 feet of stalk was visible.

4. *Natural Transmission.*

In the field gumming is transmitted naturally from one cane plant to another under suitable conditions (*see* page 41). It was thought possible that the same agencies might be active in transmitting the disease from corn to cane, and *vice versa,* and with that idea in mind two trials were laid out. Both were planned on the same lines, viz., setts of the cane variety S.J. 4—a variety very susceptible to gumming—were planted in rows the customary 4 feet 6 inches apart and good germinating conditions maintained. Approximately six weeks later, after the cane had germinated, corn was planted in single line plots of about six hills each between the rows of S.J. 4. The later planting of the corn was necessary to ensure a large cane leaf area being exposed while the corn plants were growing and green-leafed, and also ensure a mingling of the cane and the corn leaves at more or less equal heights. The plants were irrigated from an overhead spray installation, and were not artificially protected from the wind, so that conditions should have been reasonably favourable at various times for the spread of gumming disease. In one trial with both sweet and dent varieties the cane was inoculated by the usual method; in the other, the corn (sweet varieties only) was inoculated by the method 2 (*a*) outlined above, except that a single culture and not a mixture of cultures was used in preparing the inoculum. This culture had been isolated from corn artificially inoculated in an earlier experiment, and inoculation tests into cane leaves had proved its pathogenicity to cane.

All inoculations made (both natural and artificial) are tabulated in Table I.

Results.

The results obtained from the various series of inoculations are set out seriatim below.

Corn.

Method 1 (*a*) (Heart Inoculation—needle puncture).—The plants were growing rapidly, and within five days inoculation wounds could be seen on the expanded leaf blades. All wounds were small, with a halo of dead tissue, and in many cases a single white vascular bundle ran for a short distance from the puncture. Short, diffuse chlorotic streaks were also associated with the needle punctures in both inoculated and control plants. On the seventh day definite streaks running from inoculation marks began to appear. Usually the young streak was an irregular, diffuse, water-soaked strip about a white vascular bundle, varying in width, with a maximum of 1 cm. and gradually fading as it approached the sheath or the leaf margin. Within a few days the chlorotic tissue became a little more regular in outline and the infected vascular

Table I.

SUMMARY OF INOCULATIONS.

Plant.	Common Name and Varieties.	Method of Inoculation.	Number of Inoculations.
Zea mays L.	Dent Corn: Silvermine, Fitzroy, Hickory King, Yellow Dent, Funk's 90 Day, and Star Leaming	Heart, 1a	29
Ditto	Dent Corn: Leaming, Fitzroy, and Hickory King	Heart, 1b	148
Ditto	Sweet Corn: Golden Bantam, Early Cory, and Crosby's Early	Heart, 1b	29
Ditto	Sweet Corn: Golden Bantam, Early Cory, Crosby's Early, Stoke's Vanguard, No. 77 U.S.A., Howling Mob, Golden Sunshine, Gold Coin, Honey June, Red-Green, Bantam Evergreen, and Delicious	Leaf, 2a	176
Ditto	Dent Corn: Leaming and Fitzroy ..	Leaf, 2a	48
Ditto	Sweet Corn: Golden Bantam, Early Cory, and Crosby's Early	Leaf, 2b	71
Ditto	Dent Corn: Leaming and Fitzroy ..	Leaf, 2b	48
Ditto	Dent Corn: Leaming and Fitzroy ..	Stem or Cob Shank, 3	18
Ditto	Dent Corn: Star Leaming, Fitzroy, and Hickory King	Natural, 4	1 Trial
Ditto	Sweet Corn: No. 77 U.S.A., Bantam Evergreen, Gold Coin, Stoke's Vanguard, Red-Green, Howling Mob, and Delicious	Natural, 4	2 Trials
Sorghum spp. Snow. ..	Grain Sorghum: No. 1501, No. 1882, Feterita, and Broom Millet	Heart, 1a	16
Ditto	Sweet Sorghum: Imphee	Heart, 1a	4
Ditto	Grain Sorghum: Hegari, Natal 6, Dwarf Pink, Brown Yolo, Day Milo, Kalo, Wheatland Milo, American Early Red, and Sharon	Heart, 1b	262
Ditto	Sweet Sorghum: Italian	Heart, 1b	18
Sorghum verticilliflorum Stapf.	Bastard Sorghum	Heart, 1b	55
Sorghum sudanense Stapf.	Soudan Grass	Heart, 1b	61
Panicum maximum Jacq.	Guinea Grass	Heart, 1b	59
Brachiaria mutica (Forsk.) Stapf.	Para Grass	Heart, 1b	32
Pennisetum purpureum Schum.	Elephant Grass	Heart, 1b	15
Sorghum halepense (L.) Pers.	Johnson Grass	Heart, 1b	17

bundles turned brown or brownish pink, while the length of streaks increased to as much as 25 cm. Fifteen days after inoculation the streaks had further increased in length, and developed small pale red-brown spots which rapidly enlarged to form semi-circular red-brown areas up to 1 cm. diameter, with the base on the discoloured vein. The streaks had not developed further when the aged leaves were shed. *B. vasculorum* was easily isolated from points on the streaks up to 27 cm. from the infection centre. At approximate maturity stalks which had borne infected leaves were cut and sweated, but no exudation of gum occurred. Control shoots showed some physical damage to the tissues in the vicinity of the points of needle insertion, but no streaks developed therefrom.

FIG. 3.—Leaf streaks produced following heart inoculation of Fitzroy variety of corn. From left to right: Leaf showing inoculation wounds (see towards base) and young narrow streak; broad diffused streak showing initial necrosis (towards top); complete necrosis of streak.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Within seven days after inoculation the wounds caused by the entry of the needle were exposed on the young leaf blades issuing from the spindle. In the controls and in all sweet corns they were small and resembled those obtained from needle puncture inoculation of the heart (1 (*a*) above). The dent corns (Fitzroy and Leaming) showed extensive damage, the wounds being very much enlarged, up to 2-3 mm. wide and 6 cm. long, with the long axis parallel to the veins of the blade. Streaks (*see* Fig. 3) became visible on the seventh day in both dent

and sweet corns; practically all arose from the point of inoculation. At first these were irregular strips of whitish tissue about a whitened vascular bundle, but a few days later the infected veins reddened and small patches of dead tissue developed at various points in the streak. These patches rapidly spread in the dent corns, and eventually ran together to form pale, brown stripes of necrotic tissue edged with dirty white. In the case of the sweet corns, however, development of streaks did not proceed beyond the stage described in 1 (*a*)

FIG. 4.—Malformed top and tassel following heart inoculation of corn variety Leaming. Note damage to and streaks on younger leaves.

above, nor could gum be sweated from the stems at maturity, which was reached some two months later. It should be noted here that the sweet corns, unlike the dent corns, tasselled shortly after inoculation.

In the dent corns, the streaks were very numerous, up to 30 streaks appearing on the six or eight leaves of one shoot. Streaks appeared on leaves and portions of leaves not showing inoculation marks, but generally the two were associated. All streaks developed large, regular necrotic areas up to 10 cm. long by 0·5 cm. wide. There was no trace of the semi-circular areas described above (1 (*a*)) and by Ivanoff [**11**, 1935]. There was little colour in the streaks and the pale-brown dead tissues merged directly into the white of the rest of the streak. Gum was sweated from cross sections of such streaks on turgid leaves.

The areas of necrotic tissue increased during the next few days, often involving the whole streak and fusing to form necrotic bands up to 2 cm. wide. Generally, the younger leaves (i.e., those formed after inoculation) were free of symptoms, but in about 10 per cent. of the stalks, streaked, chlorotic and wilted leaves developed and the folded spindle readily yielded *B. vasculorum* in culture. Four weeks after inoculation several stalks had died, and, on

Fig. 5.—Extreme case of malformation of top of stalk of corn variety Fitzroy; eight weeks after heart inoculation.

sectioning, gum cavities were found right down to the base of the stem. Transverse sections throughout the length of the stem sweated gum profusely.

Seven weeks after inoculation most of the infected plants and all controls had tasselled and cobbed normally, but the remaining damaged shoots, after appearing generally unthrifty for some weeks, developed malformed tops, with or without the death of the growing point (*see* Figs. 4 and 5). Stalks, leaves, cobs and tassels were involved, and the upward growth of the plants ceased. Gum cavities were present in stems and/or cob shanks, and were

particularly large in the central portion of the shanks. Many of the cobs sweated gum from sections cut throughout their length and gum was occasionally present in large amounts between the cob and sheath. The few seeds set on the damaged cobs failed to germinate and attempts to isolate *B. vasculorum* from surface sterilized seeds were unsuccessful.

FIG. 6.—Streaks passing down from leaf blade to sheath of corn variety Fitzroy.

Six or seven weeks after inoculation many streaks in both distorted and apparently healthy normal plants had passed down into the leaf sheath (Fig. 6), where they were more irregular, more diffuse and ill-defined than in the blade. When sectioned and placed in a closed container these sheath streaks sweated gum copiously.

All inoculated stalks were sweated when the cobs were approaching maturity. Gum oozed from stems or cobs of more than 30 per cent., including

plants which, after showing numerous streaks, had produced normal leaves, tassels and cobs. The drying-out, attendant upon maturity, may have precluded the exudation of gum from many of the stalks which failed to yield positive results. Gum cavities were not usual in plants which had developed normally.

Method 2 (*a*) (Needle Puncture of Leaves).—Of some 176 inoculations made into sweet corn, only 30 streaks eventuated, but the hot, dry weather experienced during one test may have militated against positive results. These streaks resembled those obtained following heart puncture inoculation (1 (*a*)). No further symptoms developed, the plants tasselled and cobbed normally, and no gum could be sweated from the stems. The 48 inoculations into the dent corn varieties did not yield any results, but dry atmospheric conditions did not favour leaf inoculation.

Method 2 (*b*) (Needle Scratching of Leaves).—No infection was obtained by this method, but again unfavourable weather conditions may have been responsible.

Method 3 (Hypodermic Inoculation in Stem or Cob-shank).—Absolutely no external pathological symptoms appeared at any time, but when the sticks were cut and sweated at maturity some sixteen weeks after inoculation, several oozed gum. No significance can be attached to the proportion of sticks oozing gum, since they were dry or drying out when cut. Seed from cobs from these stalks germinated satisfactorily and no obviously diseased plants were produced. *B. vasculorum* could not be isolated from a dozen selected seedlings, although numerous yellowish, slimy colonies of a rapidly growing bacterium appeared on the dilution plates. Inoculation of this bacterium into cane gave negative results.

Method 4 (Natural Transmission).—Inoculation of the corn yielded streaks of the type which has been described under needle inoculation of leaves (2 (*a*) above), and although the leaves of S.J. 4 were in close contact with the diseased corn for a period of twelve weeks no symptoms appeared on the cane. The inoculated S.J. 4 developed numerous typical streaks but no other symptoms during the course of the trial. The corn planted between the rows of cane remained free from infection, except for the variety Fitzroy. Three streaks appeared on different plants of this variety, and yielded *B. vasculorum* from isolations. The streaks were similar to those obtained by needle puncture inoculation into the heart, and appeared to have had their origin in small holes which may have been of insect origin.

Sorghum.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Symptoms developed in all varieties of sorghum inoculated, although some were more severely affected than others.

Inoculation wounds, when exposed on the leaf blades, were usually somewhat enlarged, up to 2 mm. in diameter, and were edged with mottled dark or light-red areas which sometimes fused to form a band across the leaf. In many shoots the discoloured band occurred a few centimetres above the wounds

(*see* Fig. 7). It is suggested that the method of inoculation into the tightly rolled spindle may have forced the inoculum above the actual point of entry of the needle. A similar condition was observed following inoculation of the other *Sorghum* species. Wounds caused in mature leaf sheaths by the passage of the needle were generally surrounded by a dull-red discoloration, but did not usually give rise to streaks.

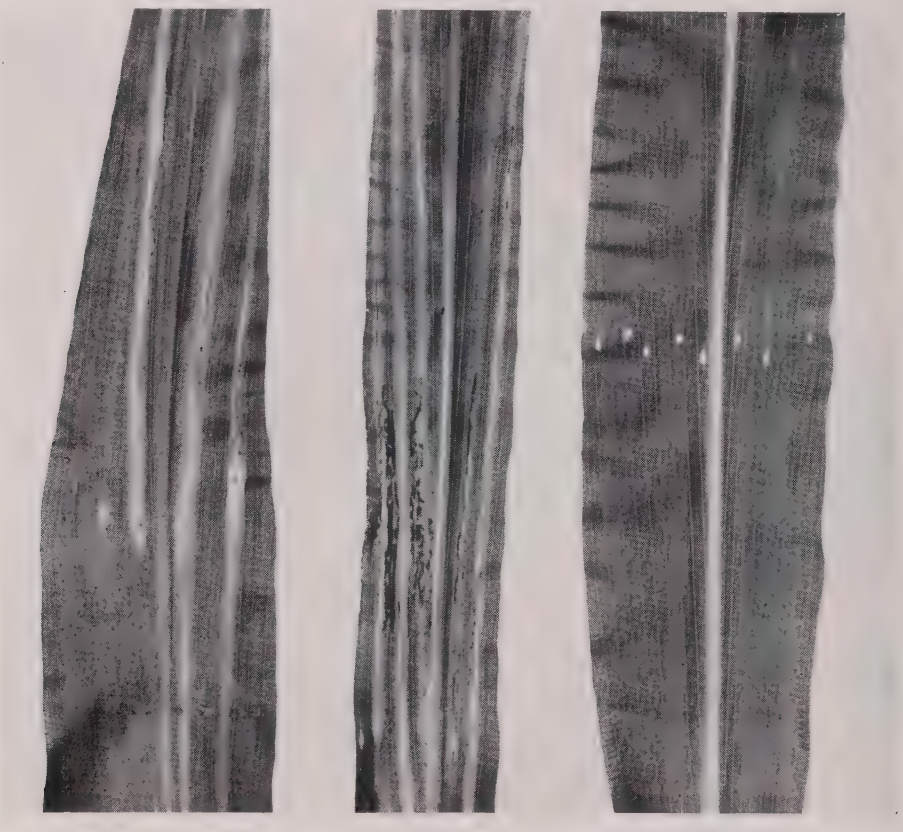

FIG. 7.—*Sorghum* species. Left to right: Leaf streaks arising from needle puncture wounds, note red dots on streaks, discoloured band above inoculation wounds and inoculation wounds in control series.

Control shoots (*see* Fig. 7) showed enlarged holes on the leaf-blades, but they healed normally and nothing further developed.

Numerous leaf-blade streaks (*see* Figs. 7 and 8) running from wounds, appeared within five to nine days; the first symptoms were usually either narrow discoloured areas, markedly speckled with red, lying about red or red and white vascular bundles, or regular chlorotic streaks with numerous diffuse red dots. Streaks were often almost as long as the blade, and up to 5 mm. in width, although fusion of adjacent streaks frequently involved a larger area;

extensions onto the leaf sheaths and midribs were irregular and dark red in colour. In later stages necrosis within the streaks developed reddish areas consisting of long narrow stripes, or a series of semi-circular spots along the infected bundle; alternatively the whole streak became brown, edged with red. This red discoloration varied from a bright, brick red to a dull, almost purple, shade, depending on the variety.

FIG. 8.—*Sorghum* species. Young leaf streaks showing early necrosis.

In some instances inflorescences on diseased plants showed dark-red blotches on the panicle branches and flower glumes. Wilting, chlorosis and red dotting of young leaves, marked stunting, extensive gum pockets in the stem and death of the growing point were associated with the systemic infection developed in many plants. The first positive signs of systemic infection were observed about four weeks after inoculation. The cessation of growth in the main axis of many badly infected plants caused the shooting of buds situated below the region of gum cavities. These side-shoots bore no symptoms of gumming disease, and some flowered normally.

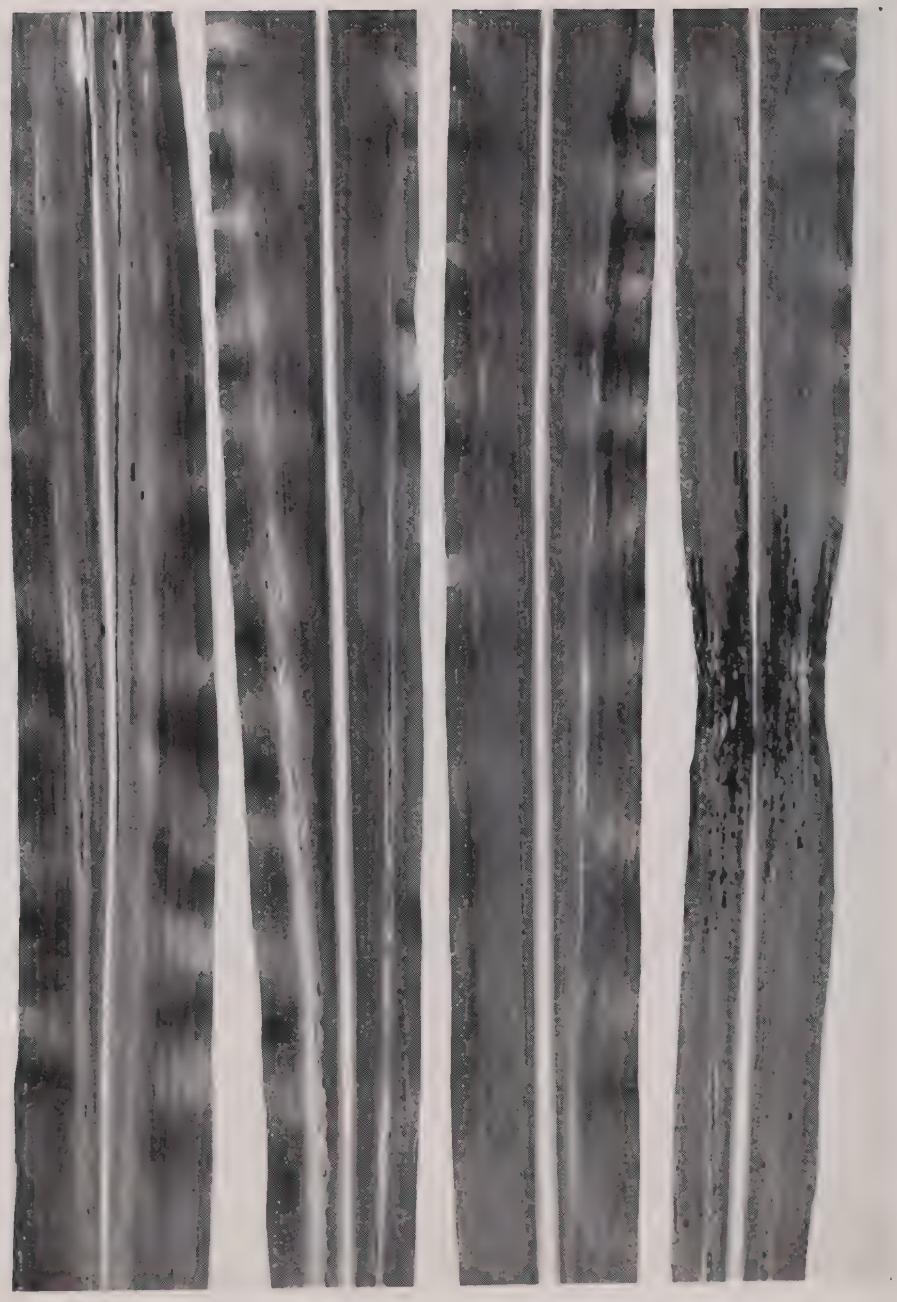

FIG. 9.—*Sorghum verticilliflorum.* Leaf streaks showing different stages of development; note red dots on streaks. The right-hand figure illustrates the development of a red mottled band about the point of inoculation.

Sorghum verticilliflorum.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Inoculation wounds appeared on the leaf blades within three days, and showed pronounced red or red and white borders up to 2 mm. in diameter, with thin, dark-red streamers, up to 6 cm. long, running down the veins. The fusion of adjacent borders often produced a mottled band (*see* Fig. 9), 2 cm. deep, across the leaf, while in a number of instances similar bands developed a few centimetres above the inoculation wounds. *B. vasculorum* was easily isolated from these areas. Wounds in controls were generally bordered by a thin white halo, and healed normally.

Streaks appeared from six to ten days after inoculation, and commenced either at a wound or in the undamaged tissues a few centimetres below. They frequently ran the full length of the blade, were 2-4 mm. wide, fairly regular in outline, and chlorotic but usually flecked with numerous red dots and red-bordered small brown necrotic areas concentrated about a reddened vascular bundle.

Further symptoms did not develop and all shoots subsequently flowered normally.

Soudan Grass.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Three days after inoculation the growth of the young leaves made the needle punctures visible. In the inoculated shoots, the wounds, enlarged up to 2 mm. in diameter, were each surrounded by a small dark-red halo, a number of which, in many leaves, fused to form a band 3 cm. deep across the leaf. Vascular bundles running therefrom were reddened for distances up to 2 cm., and were bordered by narrow zones of irregular water-soaked tissue. The wound margins in the controls were brown or white rather than red, and no reddened bundles were observed. White bundles occurred in a few instances in both control and inoculated plants.

Three days later definite streaks, generally running from inoculation wounds, and often extending to the tip of the blade, had developed on a number of shoots. Typically, a streak consisted of a fairly regular white to yellow strip, 1 mm. to 4 mm. wide, enclosing a reddened or partly reddened vascular bundle, and dotted with small diffuse red areas resembling the spots in gumming disease in cane. Two weeks after inoculation, the mature streaks had developed irregular red-bordered, brownish-red necrotic areas up to 10 cm. long and 1 cm. wide. Gum could be sweated from the freshly cut cross sections of streaks, and plantings of streak tissue on Wilbrink's medium yielded colonies of *B. vasculorum*. Occasional streaks developed on leaves which showed no inoculation wounds, but it is possible that wounds occurred on the edges of the leaves and therefore could not be definitely identified. No such streaks or reddening developed in the controls, although occasionally a diffuse water-soaked streak ran from the inoculation mark for a distance of 4-5 cm., but no organism could be isolated from these. Inoculation wounds remained small and inconspicuous in all controls.

FIG. 10.—Progressive stages in development of leaf streaks and necrosis following inoculation of *Brachiaria mutica* (Para grass).

Streaks were the only symptoms produced in the inoculated plants, and gum could not be sweated from the stems. All shoots flowered normally.

Johnson Grass.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Stools were poorly grown and shoots small, so that any symptoms developed in this instance might not be characteristic of this grass. Control shoots showed needle punctures in the leaves and nothing further developed. Wounds on the inoculated leaf blades were generally inconspicuous, although occasionally bordered with red. Streaks, resembling those described for *Sorghum verticilliflorum,* but somewhat narrower, appeared in a few leaves showing inoculation wounds, and these yielded *B. vasculorum* by isolation. No other symptoms developed.

Para Grass.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Inoculation wounds on the leaf blades in both control and inoculated shoots were enlarged and surrounded by narrow white haloes. These wounds did not show any alteration during the life of the leaves except when enveloped in the necrosis of an old streak. Young streaks, consisting of irregular, faint, diffuse, chlorotic beadings about slightly whitened vascular bundles, developed within six days after inoculation. The majority of the streaks, although still irregular and generally fragmented, became more pronounced five or six days later, and developed small, pale-brown, necrotic spots and occasional similar larger areas. Streaks were about the full length of the blade (*see* Fig. 10), up to 3-4 mm. in width, and nearly all originated at inoculation wounds. At this stage the irregular and diffuse white patches strongly suggested the symptoms of chlorotic streak disease in sugar cane [**2**, 1929]. Extensive light-brown, dead patches eventually involved practically the whole streak so that the original irregular outline was lost, except at the extremities.

New growth did not show leaf symptoms, and all stems failed to ooze gum when sweated in a warm, moist chamber over night.

Elephant Grass.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Inoculation wounds subsequently exposed on the leaf blades were enlarged but not discoloured; faint, diffuse, whitish streaks, about whitened vascular bundles extending from these wounds were observed six days after inoculation. Within a few days the streaks broadened irregularly, became more nearly chlorotic and developed small, pale, necrotic spots in the parenchyma. The mature streaks often extended the full length of the blade, were 3-4 mm. wide, usually fragmented, and consisted of whitened areas enclosing irregular, pale, brownish-pink, dead tissue (*see* Fig. 11). The streaks somewhat resembled those produced on Para grass, except that they were much longer. Further symptoms did not develop, and all shoots flowered normally.

FIG. 11.—Leaf streaks of fragmented type produced after heart inoculation of elephant grass.

Guinea Grass.

Method 1 (*b*) (Heart Inoculation—hypodermic needle).—Guinea grass was the only inoculated grass which failed to become infected. Inoculation wounds on the leaf blades were small and inconspicuous and healed normally in all shoots. Occasionally, vascular bundles, which had been pierced by the needle, showed slight chlorosis, but *B. vasculorum* could not be cultured therefrom.

DISCUSSION.

Suitable inoculation of susceptible varieties of sugar cane with *B. vasculorum* produces characteristic leaf streaks. Such may be up to 6 mm. wide, but are usually less and vary in length from a few millimetres to almost the full length of the blade. They are straight, fairly regular in outline and uniform in width, more yellow than white in colour and are usually flecked with a large number of small red blotches. As the streaks age necrotic areas appear at the point of inoculation and spread till most of the streak is involved. The streaks may extend into the leaf sheaths, but are faint and ill-defined there, and usually without necrotic areas. The development of streaks on the leaves may be followed by systemic infection, resulting in chlorosis, wilting, death of the growing point and formation of gum pockets in the stem.

Simple needle heart inoculation in corn led to the production of streaks usually about 4 mm. wide with a maximum of 1 cm., whitish in colour and irregular in outline. This local chlorosis was centred about veins which were often white or reddish-brown in colour. Small discrete necrotic areas, with the long axis running in the direction of that of the leaf, developed at irregular intervals in the streaks. No other symptoms were observed in the varieties inoculated. These streaks resemble those described by Ivanoff [**11**, 1935], as resulting from inoculation of *B. vasculorum* into sweet corn.

Mass inoculation of the organism into the heart of dent corn plants by means of an hypodermic needle produced more severe symptoms. The resultant streaks in the early stages resembled those described immediately above, but within a few days the necrotic areas spread and fused lengthwise to form a strip of irregular outline. The width of the streaks ranged up to 6 mm., although fusion of adjacent streaks involved a greater area of leaf tissue. The streaks were not so marked in the leaf sheaths and usually bore only odd patches of dead tissue in a discoloured strip. Systemic infection followed in many cases, and chlorosis of young leaves, death of the growing point, general distortion of the top and gum pockets in the stem resulted. The causal organism was isolated from the stems, leaves and cob shanks of infected plants, and under favourable conditions gum could easily be sweated from those organs.

Streaks in corn, allowing for the somewhat shorter leaf, were quite as long as those seen in cane, but appeared to differ in that the affected parenchyma

first assumed a white rather than a yellow colour; the small red dots characteristic of the cane infection were absent, and the outline, particularly of the young streak, was more irregular. The incubation period was very much shorter in corn; it was usually less than fourteen days and often less than ten, while in cane it is rarely less than fourteen days; this may possibly be correlated with the more rapid growth of corn. As in cane, the corn plant can appear healthy while carrying the disease organism within its tissues.

These results confirm Ivanoff's [**11**, 1935] findings regarding the ability of *B. vasculorum* to live in the tissues of sweet corn plants, although symptoms expressed were somewhat dissimilar from those described by him. Ivanoff reported a rather severe systemic infection, whereas the inoculations here described produced only leaf streaks in sweet corns. The reason for the dissimilarity may well be explained by different methods of inoculation, different varieties, use of different cultures or different environmental conditions ruling at the time of the respective experiments. With respect to the environmental conditions, Ivanoff apparently maintained the inoculated plants in the greenhouse, while in the present instance plants were grown in the field. Ivanoff was working primarily with *P. stewarti* when he carried out the experiments with *B. vasculorum,* and only sweet corns, known to be susceptible to Stewart's wilt, were inoculated with the gumming organism. Experiments were not made with dent corns. Although Orian [**15**, 1937] is reported to have inoculated corn plants the reviewer of that author's article does not specify the type of corn inoculated. The experiments detailed in this paper, then, are the only recorded inoculations into dent corns, which, incidentally, are the only varieties grown on a commercial scale in the Queensland sugar areas, and form practically the only cultivated crop at present likely to be of importance from the gumming alternate host point of view. The dent corns were easily infected, and *B. vasculorum* thrived so well that extensive damage resulted. The suitability of dent corn plants as hosts for the disease is also emphasised by the fact that infection was obtained naturally from gum-infected cane grown nearby.

Symptoms developed in all varieties of the sweet and grain sorghums inoculated. Needle wounds subsequently exposed on the leaf blades were usually somewhat enlarged, up to 2 mm. in diameter, and were edged with mottled dark or light-red areas which sometimes fused to form a band across the leaf. In many shoots similar discoloured bands occurred above the inoculation wounds. Streaks appeared within five to nine days, and usually first showed as either a narrow cream area speckled with red dots, about a red or red and white vein, or as regular chlorotic streaks with numerous diffuse red dots. Streaks were often as long as the blade, up to 5 mm. in width, and extended into the leaf sheaths and midribs, where they were irregular and dark red in colour. Later, long, narrow, red necrotic areas developed about the infected vein, or the whole streak became necrotic and brown, edged with dark red, although occasional streaks showed semi-circular, dead areas with the base running parallel to the long axis. Fusion of adjacent streaks often

involved a large proportion of the blade. In some instances inflorescences coming away through diseased leaves showed red blotches on both panicle branches and flower glumes. Wilting, chlorosis and red dotting of young leaves, marked stunting, extensive gum pockets in the stem and death of the growing point were, in one shoot or another, associated with the systemic infection which developed in many plants.

Inoculation wounds appeared within three days on the leaf blades of *Sorghum verticilliflorum* and developed pronounced red, or red and white areas up to 2 mm. in diameter, with thin, dark-red streamers, up to 6 cm. long, running down the veins. In a number of plants an extensive mottled red area developed a few centimetres above the inoculation wounds (*cf.* sorghum). Streaks appeared from six to ten days after inoculation, and commenced at a needle puncture wound or just below it. They were fairly regular in outline, 2-4 mm. wide, often extended the full length of the blade and usually consisted of a chlorotic area in which numerous small red dots, and small necrotic brown areas edged with dark-red, developed about a reddened vascular bundle. Further symptoms did not develop.

Streaks resulting from inoculation of Soudan grass resembled those produced in *S. verticilliflorum*. Systemic infection did not develop and the stalks failed to sweat gum.

In Johnson grass, streaks resembled those observed in *Sorghum verticilliflorum*, and there were no other symptoms.

In *Brachiaria mutica* (Para grass), streaks consisting of an irregular, faint, diffuse, chlorotic beading about a slightly whitened vascular bundle, developed within six days after inoculation. Later, the majority of the streaks, although still irregular and generally fragmented, became more pronounced and developed small pale-brown necrotic spots which occasionally enlarged considerably. Streaks extended almost the full length of the blade, were up to 3-4 mm. wide and nearly all originated in inoculation wounds. Extensive, dead, light-brown patches eventually involved practically the whole streak. There were no other symptoms, and stems failed to sweat gum.

Inoculation wounds in elephant grass enlarged but did not discolour, and six days after inoculation faint, diffuse, whitish streaks appeared about whitened vascular bundles. Within a few days the streaks broadened irregularly, became more pronounced, and small, pale, necrotic spots developed in the parenchyma. The mature streaks varied in extent, sometimes running the full length of the blade, and being 3-4 mm. wide; they were usually fragmented and consisted of whitened areas enclosing irregular, pale, brownish-pink dead tissue. Further symptoms did not develop.

It is of interest to note that virtually all species so far inoculated with *B. vasculorum* in Queensland or the United States have been found to act as hosts for that organism. These results suggest that there might exist a large number of Gramineæ, both cultivated and wild, which will be found to be

susceptible. Orian's [**15**, 1937] recent isolation of *B. vasculorum* from palms extends the host range of *B. vasculorum* beyond the confines of the family Gramineæ and opens a wide field for investigation.

The fact that alternate hosts do exist and that their number might be far greater than we are at present aware has an important bearing on the ultimate control of gumming in Queensland. There is no authenticated instance in this State of the disease having been completely eradicated from any one area—as tested by a subsequent return to cropping of susceptible varieties. The reason for this may lie in the characteristic small farm system of cane culture. When hundreds of farmers grow cane in any one district it is obviously impracticable to be certain that varieties susceptible to gumming are entirely eliminated or that the disease is not present in fields of near-resistant varieties. In short, while the non-eradication of gumming from an area may be dependent upon its continued existence in cane, this work clearly indicates that the possibilities of its survival in alternate hosts must not be overlooked. Concomitants of small farms are small fields, leading to large areas of headland, horse paddocks, diversified crops, home gardens and innumerable roadways, all contributory factors enabling possible alternate hosts to exist in close proximity to the cane. Further possible harbourage is provided by the scrub and forest still standing alongside many fields.

The resistance of a cane variety to gumming varies in different parts of Queensland (*see* page 36), so that it might be expected that the resistance of alternate hosts will also vary. Variations in susceptibility of alternate hosts from one district to another, and the narrow climatic ranges of some species, might permit different plants to act as alternate hosts in different areas. Variations in incidence of the disease in possible alternate hosts from season to season and year to year might occur, as it does in cane, so that in certain seasons these hosts may be completely free of the disease and only become reinfected from neighbouring cane, or, on the other hand, survival of *B. vasculorum* in alternate hosts may result from systemic infection of perennial stools or infection of the seed.

Although gumming disease, in the same varieties, is more serious in the South than in the North, there is no evidence that natural transmission of the disease from cane plant to cane plant is any less efficient in the North, an observation which implies that alternate hosts could be just as important there as in the South, and that transmission trials from cane to other plants and vice-versa should not be neglected in any district.

As yet there is no record of the natural occurrence of *B. vasculorum* in plants other than sugar cane, with the exception of the white palm Orian [**15**, 1937]. However, the facility with which positive results have been obtained upon inoculation of the selected experimental plants and, relative to sugar cane, the atypical nature of the symptoms produced in most cases, both suggest caution; and accordingly we feel that a policy of widespread replanting to highly susceptible varieties in a once-infested area is not justifiable at this juncture.

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